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CASE REPORT

An unusual complication of gallstones: extrinsic compression of the portal vein leading to liver hypoperfusion

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Introduction

Gallstones are common, with many patients remaining asymptomatic. Clinical manifestations of gallstones include biliary colic, cholecystitis and obstructive jaundice.

Case report

We present a patient with an unusual radiological finding discovered during the investigation of right upper quadrant pain.

A previously fit and healthy 64-year-old lady presented to the outpatient clinic with a 6-month history of colicky right upper quadrant pain. There were no other associated symptoms. The patient had no recent weight loss, no past history of note and was not taking any medication. On examination there was a tender palpable right upper quadrant mass. This was clinically thought to be an enlarged tender gallbladder. All routine blood tests were within the normal ranges except the GGT, which was mildly elevated at 52 (normal = 7–33 IU/l). Ultrasound showed a liver normal in size and appearance, and a distended gallbladder measuring 12 cm in length, which contained gallstones and sludge (Fig. 1). The largest stone measured 3.4 cm. The common bile duct measured 6.7 mm (normal <6 mm).

Contrast CT was performed to exclude alternative causes of a right upper quadrant mass. This too showed a distended gallbladder with a large calculus impacted within the neck, raising the possibility of a mucocoele. In addition, an unusual appearance of the liver was noted on the post-contrast study (portal venous phase of contrast enhancement). On the pre-contrast images the liver showed uniform attenuation (Fig. 2), but on the post-contrast images there was a large wedge-shaped area of low attenuation within the right lobe of the liver (Fig. 3). The possibility of impaired perfusion was raised. The portal vein was noted to be normal within the porta hepatis, but the calculus within the gallbladder neck was seen in close proximity to the right branch of the portal vein, possibly compressing it.

MRI of the abdomen and an MRCP (magnetic resonance cholangiopancreatogram) were performed. These confirmed that the gallstone within the gallbladder neck had caused an upward displacement and compression of the right main branch of the portal vein (Fig. 4). No gallstones were seen within the common hepatic or common bile ducts. The extrahepatic biliary tree was of normal calibre.

The mucocoele was drained percutaneously under ultrasound guidance to facilitate disimpaction of the gallstone and restore portal vein perfusion. The initial material drained was mucopurulent, followed by clear bile, indicating that the stone had disimpacted and there was now a patent cystic duct. Post-procedure contrast CT of the liver in arterial and portal venous phases of contrast enhancement appeared normal (Fig. 5), confirming that the previous vascular hypoperfusion was induced by the calculus.

The patient underwent an uncomplicated laparoscopic cholecystectomy. There were no unusual anatomical variations. The gallstones were retrieved from the gallbladder. Histology reported a gallbladder with changes consistent with chronic cholecystitis. The patient made an uneventful

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Figure 1 Ultrasound of the abdomen showing large gallstones and mucocoele.

recovery and was free of symptoms at the review clinic 6 weeks later.

Discussion

Gallstones are common and not always associated with any clinical problems. This was a case of chronic cholecystitis, an impacted gallstone leading to a mucocoele, and compression of the right main branch of the portal vein leading to hypoperfusion of the liver. To our knowledge this has not been previously described, perhaps because we do not routinely investigate patients with symptomatic gallstones using contrast-enhanced CT.

Is there any clinical relevance to the finding? Would the patient have developed hepatic symptoms? Perhaps not, but in this case discovering the portal vein compression did change our management. A decision was made to drain the mucocoele

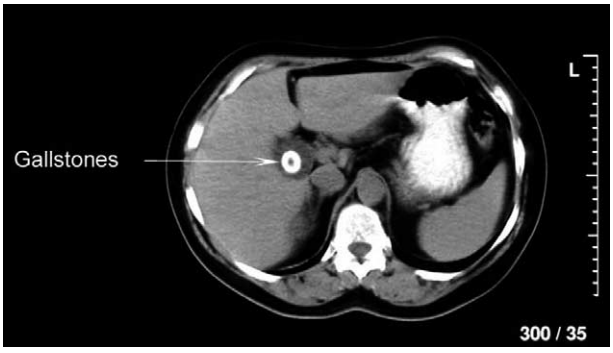

Figure 2 Pre-contrast CT showing normal liver.

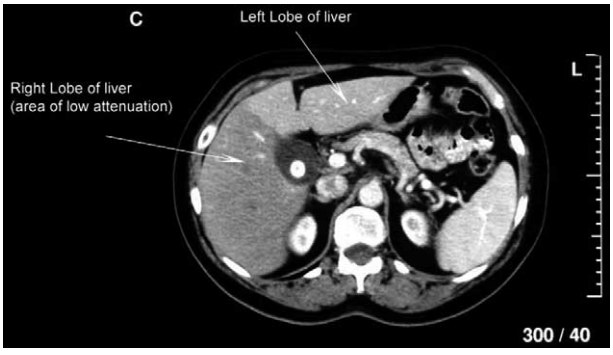

Figure 3 Post-contrast CT showing area of low attenuation in right lobe of liver.

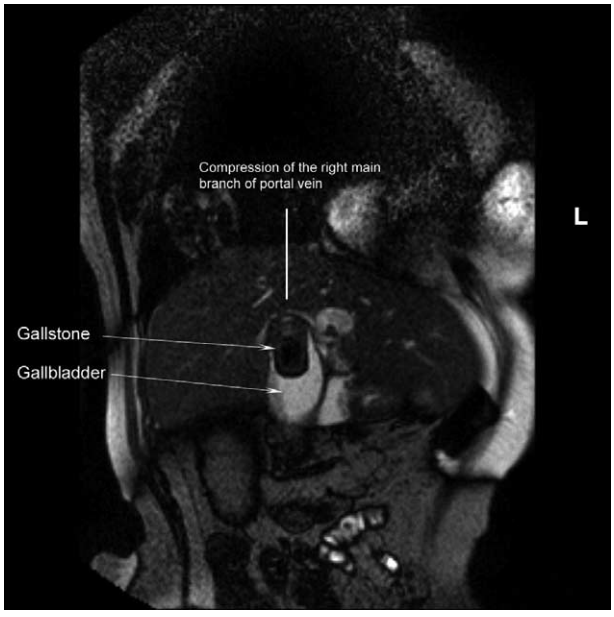

Figure 4 Oblique coronal MRI showing compression of right main branch of portal vein.

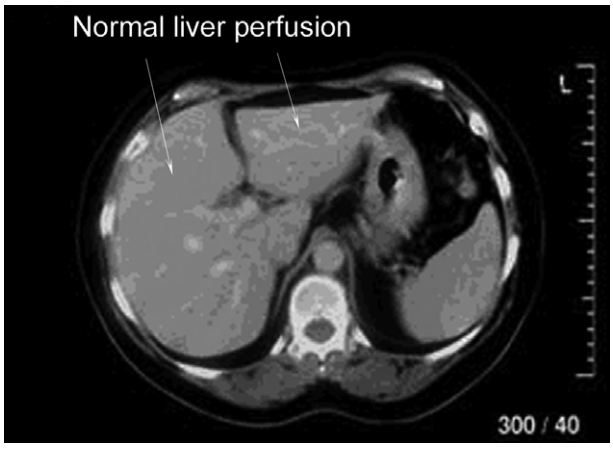

Figure 5 Post-contrast CT following drainage of mucocoele and dislodgement of gallstone from gallbladder neck, showing normal liver perfusion.

before laparoscopic cholecystectomy. The resulting disimpaction of the gallstone, with a subsequent normal CT study, reassured us that no other hepatic pathology was present.

In the literature a number of causes have been suggested of hepatic attenuation differences on contrast-enhanced CT. Transient hepatic attenuation differences have been reported in 9.3% of dual-phased contrast-enhanced CT studies performed,[6] the most common cause being chronic cholecystitis (40%), followed by previous biliary surgery. Several papers have commented on causes of focal decreased liver densities, such as focal fatty change, hepatomas,[2] liver metastasis,[2] haemangiomas, angiolipomas,[4] lymphomas[3] and amyloidosis, but these are not necessarily wedge-shaped. Causes of diffuse homogeneous liver hypoperfusion include tumour thrombus in the portal vein,[5] fatty infiltration of the liver,[1] hepatic venous outflow obstruction (Budd–Chiari syndrome), inflammatory processes, anatomical variants in hepatic blood supply[5] and tumours compressing the portal vein, which may cause wedge-shaped portal hypoperfusion. In this particular case, all these could be excluded following drainage of the mucocoele. After the gallstone had fallen back into the gallbladder, decompressing the right portal vein, CT showed the liver to be normal.

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